90 Abdominal Gas

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Definition

The complaint of abdominal gas usually refers to chronic belching, abdominal distention (bloating), or excessive rectal flatulence, or to a combination of these symptoms.

Technique

Patients often complain of having too much gas. An essential initial step in evaluating that complaint is to ask what is meant by "too much gas." The clinician must determine whether there is excessive belching, whether bloating is present, or whether the patient is passing excessive rectal gas.

If *belching* is a problem, the patient must be questioned about the oral intake of substances known to release excessive amounts of gas in the stomach, for example, carbonated soft drinks, beer, effervescent forms of certain medications. In attempting to establish a relationship of such substances to the symptom of belching, the clinician must also determine the quantities used and the time they are used.

In regard to belching, it is equally important to consider the possibility of air swallowing, which can be associated with several factors. Excess air is often swallowed along with the excess saliva stimulated by the habitual use of such substances as lozenges, tobacco products, and chewing gum. A similar situation can occur with a chronic posterior nasal discharge. Air often enters the esophagus when food and liquids are ingested simultaneously or when large volumes of liquid are drunk in a gulping fashion.

The clinician must also consider conditions associated with mouth dryness. Such a state usually prompts the patient to carry out smacking movements in an attempt to wet the lips and oral cavity; air swallowing often accompanies such movements. Sjögren's syndrome and radiation therapy to the neck often reduce the secretion of saliva. A much more common cause of dry mouth, however, is the use of medications with anticholinergic side effects.

Air swallowing can also occur either as a habit or as a response to stress. Although the patient may not have noted whether swallowing movements precede the belching, it is worth seeking this information. In fact, during the process of taking the history, the clinician should be observing whether swallowing precedes the eructation of gas. Since air swallowing is frequently associated with stress, questions that might relate the symptom to stressful situations may be particularly useful.

By the complaint of abdominal *bloating*, the patient usually is referring to a sensation of generalized abdominal fullness. At times the patient will attempt to document this symptom by relating it to changes in the tightness of clothing or to visible changes in the size of the abdomen. At other times the symptom of fullness may be present without any visible changes. Questioning should determine whether the symptom occurs daily or only occasionally; whether it can be related to ingestion of certain substances; and whether the symptom is relieved by belching, by passing rectal gas, or by using medications.

With regard to excessive rectal *flatulence*, several questions may be helpful. Has the patient noted the association of the symptom with any particular food substances? Is there a relationship of rectal gas to the consumption of milk or milk products? If so, does this apply also to yogurt or cream? In this regard, information regarding the patient's ethnic background may be particularly useful. Is the patient of Northern or Middle European extraction or do the patient's ethnic roots stem from Southern Europe, Asia, or Africa? Does the patient eat certain legumes or breads? Do the patient's stools float or sink?

Basic Science

Gas is introduced into the gastrointestinal tract in several ways. Although we are all familiar with the belch produced immediately following the drinking of a carbonated beverage, a very common antecedent to belching is air swallowing (aerophagia). Radiologically, it can be demonstrated that the swallowed air usually goes partway down the esophagus and then is eructated. Consistent with this, analysis of the eructated gas typically reveals such a belch to consist of nitrogen and oxygen in the proportions found in ambient air. Since some of the swallowed air occasionally enters the stomach, it is not uncommon for plain x-ray films of the abdomen to demonstrate a postbelch enlargement, rather than diminution, of the gastric air bubble in these cases.

When swallowed air does enter the stomach, the mixture of gases becomes altered because the vascular gastric mucosa itself will utilize some oxygen and allow oxygen to diffuse across the mucosa into the bloodstream. At the same time, carbon dioxide diffuses out of the blood into the gastric lumen. Thus, while the concentration of nitrogen in the gastric lumen remains unchanged, the concentration of oxygen decreases slightly, and the concentration of carbon dioxide increases slightly.

The composition of this gaseous mixture is further altered in the duodenum, where the bicarbonate in the biliary and pancreatic secretions reacts with the hydrochloric and fatty acids delivered from the stomach. These neutralization reactions produce large amounts of carbon dioxide and water. In fact, canine studies have demonstrated an average duodenal P_{CO_2} of 255 mm Hg in the fasting state, increasing to 500 mm Hg after feeding. This normal presence of *carbon dioxide* in the duodenum likely accounts for symptoms of postprandial bloating and fatty food intolerance in patients who have an increased sensitivity to intestinal distention. Much of the excess carbon dioxide produced in the duodenum is reabsorbed in the proximal part of the jejunum and is carried by the blood to the lungs, where it is exhaled.

Consequently, by the time the gaseous mixture reaches the colon, the partial pressure of carbon dioxide is similar to that noted in the mixture of gases that originally entered the duodenum from the stomach.

The source of rectal gas cannot be attributed primarily to air swallowing, since of the five gases that constitute over 99% of intestinal gas, only two—nitrogen and oxygen—are present in the atmosphere in appreciable amounts. The other three intestinal gases—methane, hydrogen, and carbon dioxide—result from the metabolic processes of bacterial flora in the colon.

In humans, the production of *methane* is confined to the colon, where the partial pressure of this gas reaches as high as 200 mm Hg. Much of that produced is absorbed into the portal blood for delivery to the lungs. Only one out of three adults consistently possesses the strictly anaerobic bacteria capable of producing significant quantities of methane. This capacity appears to be a familial phenomenon, unrelated to dietary factors. The offspring of two methane producers have a 95% chance of being methane producers. If only one parent produces methane, the offspring have a 50% chance of producing methane. If neither parent produces methane, there is a less than 10% chance of the production of methane by the offspring. This familial tendency appears to be environmental rather than genetic and results from colonization of the colon with methane-producing bacteria early in life, such as by contamination during the actual birthing process or by contact with other family members during infancy. Methane production becomes detectable only after the first few postnatal weeks and reaches the adult level by age 8 years. It is the incorporation of methane into fecal material that most commonly accounts for floating stools in healthy subjects. Even in patients with malabsorption, floating stools result more likely from the gas and water content than from the fat content.

The bacteria that produce *hydrogen* and *carbon dioxide* as metabolic products are much more generally distributed in the population and are similarly confined to the colon. These bacteria depend on substrates that can be fermented, such substances being mainly carbohydrates that are both indigestible and unabsorbable by the small intestine. One such substance in patients with a small intestinal lactase deficiency is lactose, a sugar in milk. Lactase deficiency may transiently occur as a result of an acute illness of the small intestine, or it may occur on a long-term basis as a result of chronic enteric disease. More commonly, long-term lactose deficiency develops sometime during the first two decades of life on an autosomal recessive basis. Thus, most people of ethnic origins other than Northern and Western European have an impaired ability to break down lactose into its absorbable components of glucose and galactose. The degree of impairment of lactose absorption in such subjects varies, reaching as high as 75% but usually being about 50%. This variation accounts for the wide range of lactose tolerance even among lactase-deficient subjects. In addition, even normal subjects fail to absorb up to 8% of a lactose load.

Interestingly, most lactase-deficient individuals tolerate yogurt, a milk product that contains the amount of lactose found in unconcentrated milk. Consistent with this observation are breath hydrogen studies that indicate that the reduced symptoms of flatulence and diarrhea with yogurt compared to milk correspond to reduced gaseous fermentation products when yogurt is used. This phenomenon can be attributed to beta-galactosidase, a lactase contributed to the yogurt by the two bacteria (*Lactobacillus bulgaricus* and *Streptococcus thermophilus*) used in its preparation. Lactase-deficient individuals also tolerate products made from cream, since lactose, being water soluble, is contained mostly in the aqueous portion of milk. Consistent with this is the poor tolerance of ice cream, which is made mostly from milk.

For most people, baked beans, soybeans, and other legumes also result in increased production of hydrogen and carbon dioxide by colonic bacteria. Although indigestible oligosaccharides have long been regarded as the main substrates in these reactions, recent evidence points to starch malabsorbed in the normal human small bowel as the major source of fermentable material following ingestion of complex carbohydrates.

These same fermentation products follow the ingestion of most flours, since they contain carbohydrate that cannot be absorbed in the small intestine; the carbohydrate thus enters the colon. Gluten appears to impair the complete absorption of starch in most flours, even in many otherwise normal people who do not have celiac disease. The one flour that is virtually completely absorbed is rice flour, which does not contain gluten.

Other foods contain substances that, being nondigestible and nonabsorbable, enter the colon where they participate in bacterial fermentation processes. Mushrooms contain the disaccharide trehalose. Prunes and raisins contain a small, nonabsorbable fatty acid. Nonabsorbable sugars such as the long-familiar saccharin and the increasingly used sorbitol, contained in many so-called sugarless gums and candies, can contribute to diarrhea and the increased production of colonic gas, particularly when taken in large quantities.

The amount of hydrogen gas produced depends not only on the type and quantity of the various substrates but also on the type of colonic flora. Bacteria vary in their capacity to convert carbohydrate to hydrogen. Furthermore, only about 10% of the hydrogen produced is actually expelled rectally. Some of the hydrogen is consumed by other colonic bacteria, a phenomenon that may partially explain unsuccessful attempts at treating flatulence with antibiotics. A large portion of the hydrogen is absorbed into the portal blood for eventual excretion by the lungs. This excretory pathway has enabled breath analysis to be used to study intestinal gas production.

Another approach to the study of intestinal gas has been a washout technique with the inert gas argon. The infusion of argon into the proximal jejunum has allowed for the washout of intestinal gas and its collection by means of a rectal tube. Analysis of this collected gas has confirmed the five major components of intestinal gas to be nitrogen, oxygen, carbon dioxide, hydrogen, and methane. Of note is that these five gases, which constitute nearly 100% of bowel gas, are all nonodorous. The human ability to detect the presence of extremely small quantities of malodorous gases, such as hydrogen sulfide, can be credited to the remarkable capacity of the olfactory sense.

These studies also demonstrate that the colon contains virtually no *oxygen*, a finding likely attributable to the consumption of oxygen by colonic bacteria. *Nitrogen* may be present in the colon in varying amounts. Some may be from swallowed air. In the colon, this nitrogen may be retained as a result of the production of methane, hydrogen, and carbon dioxide, which effectively lowers the luminal P_{N_2} and thereby prevents the absorption of swallowed nitrogen. Furthermore, such high colonic nitrogen gradients conceivably may result in the diffusion of more nitrogen into the colonic lumen.

The argon washout technique has also been used to determine the volume of gas present in the intestinal tract.

Studies of normal subjects have demonstrated that the intestine contains relatively small quantities of gas, ranging from 30 to 200 cc. Of interest is that similar small amounts of gas were frequently found in patients with the common complaint of bloating and abdominal distention. Such observations are consistent with radiologic studies that did not show any correlation between the presence or absence of bloating symptoms and the volume of gas present in the intestinal tract.

A number of patients with bloating poorly tolerated the infusion of argon into the intestinal tract, sometimes to the degree that the infusion had to be stopped. This painful response was usually associated with delayed transit of the argon through the intestine and occasionally with reflux of the infused gas from the intestine into the stomach. These observations suggest that the so-called functional symptoms of bloating and abdominal pain in many patients may be due not so much to an excessive amount of gas as to an increased sensitivity of the intestinal tract to the usual amounts of gas present.

Gas-chromatic (GC) and mass-spectrometric (MS) techniques have been utilized to analyze those volatile substances that produce fecal odor. Until recently, it had generally been accepted that skatole and indole, benzopyrrole end products of the anaerobic metabolism of amino acids by colonic microflora, primarily account for fecal odor. However, GC–odor–MS analysis has indicated that volatile methyl sulfides—methanethiol, dimethyl disulfide, and dimethyl trisulfide—are likely responsible for the malodor of feces. Hydrogen sulfide gas may have only a minimal impact on the odor, particularly considering the larger concentrations of methanethiol, the odor of which cannot be distinguished from hydrogen sulfide.

There are exogenous sources of these fecal odorants, such as cocoa, beer, coffee, and a variety of vegetables, but these play only a minimal role. The major source of fecal odor is endogenous, specifically, the metabolism of nonabsorbable food substances by enteric bacteria. Further characterization of these metabolites could conceivably contribute to controlling the odor of flatus.

Clinical Significance

The evaluation and management of the problem of abdominal gas hinges on a clear understanding of the meaning of the patient's complaint. If belching is the main problem, a clinician must determine whether there is a relationship of that symptom to a variety of possible etiologic factors. Does the eructation follow large meals or the ingestion of carbonated beverages? Does the patient constantly have a dry mouth or a chronic postnasal discharge associated with frequent swallowing efforts? Has the patient developed the habit of making swallowing movements before each belch? Are the symptoms worse during moments of stress? These are some important questions that will enable the clinician to determine the most likely cause of the symptoms and the therapeutic approach that may be the most beneficial.

Awareness that bloating may be due to increased sensitivity of the patient to the usual amounts of gas present in the intestine may help the clinician to manage the problem more appropriately, thereby avoiding ineffective therapeutic measures. It is frequently helpful for the patient and the clinician to look on such symptoms as disturbances of intestinal motility.

Finally, knowing that the bacterial flora produces colonic hydrogen and carbon dioxide, and knowing that such gas production depends on the availability of nonabsorbable and fermentable substrates, the clinician can begin to approach the problem of excessive rectal flatulence by determining the patient's ethnic background and by obtaining a dietary history. Those initial steps may allow for modifications in the patient's diet while avoiding the expense, discomfort, and inconvenience associated with more elaborate diagnostic studies, which would likely contribute very little toward the management of the problem.

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